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Effect of HeNe and Pulsed Nd:YAG Laser Irradiation on Intradental Nerve Responses to Mechanical Stimulation of Dentine

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Background and Objective: **Our study aimed to determine how lasing affected intradental nerve responses to dentine stimulation.**
Study Design/Materials and Methods: **Intradental nerve activity was recorded from canine teeth of anaesthetised ferrets. Dentine exposed at the tip of the tooth was stimulated with a glass probe. After determining baseline responses to mechanical stimulation, dentine was lased using a pulsed Nd:YAG laser at 60–150mJ/pulse and 10–30 pulses/sec (total power = 0.3–3.0 W).**
Results: **The HeNe aiming beam alone and Nd:YAG laser at 0.3 W (+ HeNe) had no effect on intradental nerve responses to dentine stimulation. Lasing at 0.6–1.5 W could either enhance or suppress intradental nerve responses. Lasing at more than or equal to 2.0 W or repeated lasing at lower intensities depressed intradental nerve responses. Lasing often induced intradental nerve firing.**
Conclusion: **HeNe lasing had no effect on intradental nerve excitability. The Nd:YAG laser could depress intradental nerve responses to dentine stimulation. Lasers Surg. Med. 26:241–249, 2000.** **© 2000 Wiley-Liss, Inc.**

Key words: dentine sensitivity; HeNe laser; hypersensitive dentine; Nd:YAG laser; tooth pulp

INTRODUCTION

Lasers are advocated for a range of dental applications, including the treatment of "hypersensitive" dentine, although some authorities have expressed concerns that laser irradiation could damage the dental pulp [1]. Renton-Harper and Midda [2] found that the pulsed Nd:YAG laser was effective in reducing dentinal hypersensitivity to air-jet stimuli. These findings were supported by Gelskey et al. [3] who reported that irradiation with a helium-neon (HeNe) laser, a pulsed Nd:YAG laser, or both, caused 58–63% reduction in dentine sensitivity to cold air and mechanical stimulation. Whitters et al. [4] reported that pulsed Nd:YAG laser irradiation of healthy incisor teeth in human subjects caused a small, but statistically significant, increase in tooth pulp pain thresholds to electric stimulation.

It has been reported that Nd:YAG laser irradiation can cause melting of dentine in vitro and closure of dentinal tubules [5,6]. Goodis and coworkers [7,8] have also shown that Nd:YAG laser irradiation can reduce dentine permeability in vitro by coagulating the proteins in the fluids inside the tubules or by occluding the tubules. Lasing has also been shown to raise the temperature inside the tooth pulp [7,9,10], which contains the nerves and blood vessels. The increase in pulp temperature varied as a function of the radiation parameters (energy, pulse frequency, and duration of exposure) and also the thickness of dentine remaining over the pulp [7,9,10]. Another important factor is whether or not the tooth in vitro contains pulp tissue [11]. White et al. [9] con-

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cluded that lasing at 1.0 W (100 mJ at 10 pps) for 10 seconds did not pose a "significant pulpal risk due to temperature rise" if the remaining dentine thickness was >1 mm. However, they did report that laser-induced temperature rises could damage pulpal tissue if the dentine thickness was <1mm.

Lasing can also modify pulpal haemodynamics [12,13] and depress intradental nerve excitability to electrical stimulation [14]. However, there is little information on how intradental nerve responsiveness to more physiological stimulation is affected by laser irradiation of dentine, as opposed to irradiation of the overlying enamel. This is an important consideration because the ultimate effects of laser irradiation on teeth depend on the nature of the laser light-tissue interactions [15]. These interactions depend on a range of factors, including the optical properties of the tissue (in particular the absorption of the incident wavelength), whether continuous or pulsed excitation is used, the pulse duration and repetition rate, and the energy/power per unit area to which the tissue is exposed. The present study aimed to determine how direct lasing of dentine would affect the responsiveness of intradental nerves to physiological stimulation of the laser-treated dentine.

MATERIALS AND METHODS

The experiments were carried out on 20 adult female ferrets (each 750–1,000 g and <1 year old). Anaesthesia was induced by intraperitoneal injection of sodium pentobarbitone (Sagatal, May & Baker Ltd, UK; 42 mg/kg) and maintained throughout the procedure by 3 mg/kg increments given as required by means of a cannula in the external jugular vein. The level of anaesthesia was monitored by testing the corneal and jaw opening reflexes. The trachea was cannulated and the animal's body temperature maintained at 37°C by a homeothermic electric blanket unit. The head was immobilised by a short metal pole fixed to the frontal bone with self-tapping screws and cold-curing acrylic resin [16]. On completion of the scheduled procedure, animals were killed by an overdose of the anaesthetic administered intravenously.

Tooth and Electrode Preparation

Intradental nerve activity was recorded from the canine teeth using Ag/AgCl dentine electrodes similar to those described previously [16,17] and shown schematically in Figure 1. The Ag/AgCl recording electrode was incorporated in a prefabricated cap, composed of a Perspex cylinder 10 mm long and 5 mm in diameter. The tooth crown was cleaned with dental prophylaxis paste. The Perspex cap was adapted to the tooth crown with cold-curing acrylic resin and fixed to the tooth with polycarboxylate cement (Bayer Dental, Leverkusen, Germany). The indifferent electrode was placed in a small cavity cut just into dentine near the cervical margin of the tooth. The cervical electrode was isolated from the gingival tissues with inlay wax and supported with polycarboxylate cement.

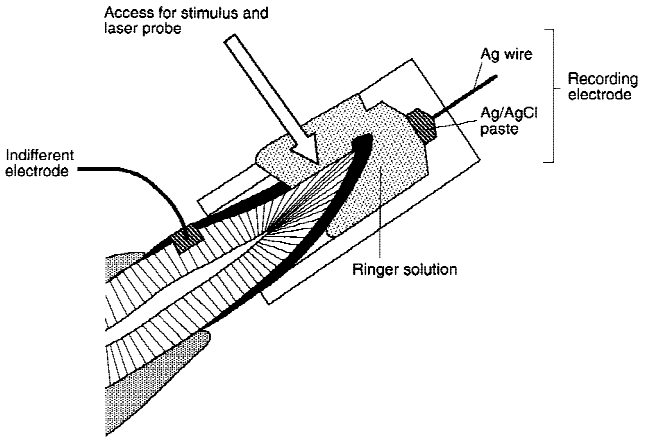

Fig. 1. A schematic diagram of the Perspex cap and electrodes in relation to the canine tooth (not to scale). Enamel (black) covers the dentine of the tooth crown. The dentinal tubules are represented by the lines extending from the inner aspect of the enamel to the pulp (shown as a white zone in the centre of the tooth).

Enamel on the lateral aspect of the tip of the tooth crown was carefully removed with a slow-running bur under Ringer's physiological saline solution until the dentine was just exposed. Cutting was continued until an area of cuspal dentine (ca. 2 mm × 1 mm) was exposed. The exposed dentine was etched with 10% citric acid solution or 35% phosphoric acid gel to remove any smear layer and then thoroughly washed with Ringer's solution. The Perspex cap was filled with Ringer's solution, which served to keep the dentine moist and also to complete the recording circuit between the Ag/AgCl electrode and the dentine. The exposed dentine was stimulated by gentle scratching with a fine glass probe introduced through an access hole in the side of the fluid-filled cap. The nonconducting glass probe made it possible to stimulate the dentine without causing electrical artefacts. In some experiments, the Ringer's solu-

tion in the cap was replaced with 1 or 2 M NaCl solutions to test responses to hypertonic solutions, which can also activate intradental nerves [16,17].

Recording and Stimulation

Signals recorded from the dentine electrodes were amplified with a Neurolog AC amplifier unit (NL 103, NL 105, NL 115 with bandpass 1 Hz to 10 kHz; Digitimer, Ltd., Welwyn Garden City, UK) and displayed on a Tektronix D11 storage oscilloscope. A combination of Neurolog modules was used to generate a ratemeter recording of intradental nerve activity. The amplifier output signal was digitized by a window discriminator (NL 200), and the digital pulses were fed to an integrator (NL 600), which was reset with a clock pulse (NL 303, NL 603) every 1 or 2 seconds as appropriate. The analogue output of the integrator was passed into an MacLab 4 interface (A.D. Instruments, East Sussex, UK) and displayed on a computer screen as a chart recorder emulation (MacLab Chart, A.D. Instruments); the processed data were stored on computer disk.

The dentine was stimulated with fine glass probes made by drawing out a glass rod in a flame. The fractured tip was passed briefly through a flame to produce a rounded end ca. 0.5 mm in diameter. Viewed with a binocular microscope, the probe tip was gently moved over the exposed dentine surface under light, steady finger pressure for periods of up to 30 seconds. The action potential discharges evoked during mechanical stimulation were quantified by using the MacLab Chart software to calculate the areas (in V.s) enclosed by the ratemeter response envelopes.

To quantify the effects of lasing on dentine sensitivity, the ratemeter data were normalised by calculating the average of the responses to probing before the laser irradiation was applied. The average was taken as the control value. The average response after lasing was then calculated and expressed as a percentage of this control level. To compensate for any changes in intradental nerve responsiveness during the experiment, the responses after a bout of lasing were always expressed in terms of the mean response immediately preceding each bout of lasing. This procedure had the advantage that it tended to mitigate the effects of occasional spurious responses but had the disadvantage of losing resolution of transient effects and trends.

Given the variations in the normalised responses to mechanical stimulation following different levels of lasing, data were initially analysed with the Kruskal-Wallis test using Minitab 8.2 software (Minitab, Inc., Pennsylvania). Where indicated, a Mann-Whitney test was used to test between group comparisons. The tests were two-tailed, and differences were considered significant when $P < 0.05$.

Lasing

The exposed dentine was irradiated by using a pulsed Nd:YAG laser (American Dental Laser dLase 300; 1,064-nm wavelength, 150-μs pulses) with a 320-μm optical fibre delivery system. A helium-neon (HeNe) aiming beam was also transmitted along the optical fibre to aid targeting of the infrared Nd:YAG laser radiation. In some control experiments, the HeNe aiming beam only was applied. The pulsed Nd:YAG laser radiation was applied using 30–150 mJ pulses at 10–30 pps (total power in the range 0.3–3.0 W) for periods of 30 seconds. During lasing, the laser optical fibre tip was moved manually to and fro over the exposed dentine at a distance of approximately 0.5 mm from the tooth surface. The fibre tip was applied under Ringer's solution, and it was possible to continue recording during lasing. The fibre optic tip was cleaved before each bout of Nd:YAG lasing. For all laser applications, the optical fibre tip was viewed with a binocular microscope to ensure that the aiming beam was directed at the exposed dentine. When the laser was in use, protective eyewear of appropriate optical density was worn in accordance with British and European safety standards [18].

RESULTS

Figure 2 shows typical intradental nerve responses to mechanical stimulation of dentine. No intradental nerve activity was recorded in the absence of dentinal stimulation. When dentine was stimulated with the probe, this elicited bursts of neural activity, which consisted of action potentials in several different sensory units within the resolution of the recording electrodes (Fig. 2A,B). When the glass probe was applied to exposed enamel at the tooth tip, no spikes were generated (Fig. 2C).

Figure 3 shows typical ratemeter outputs recorded during an experiment. The mechanical stimulus was applied continuously for 30-second periods, and the stimuli were repeated at 5-minute intervals. To illustrate how these responses were quantified, the areas of the initial

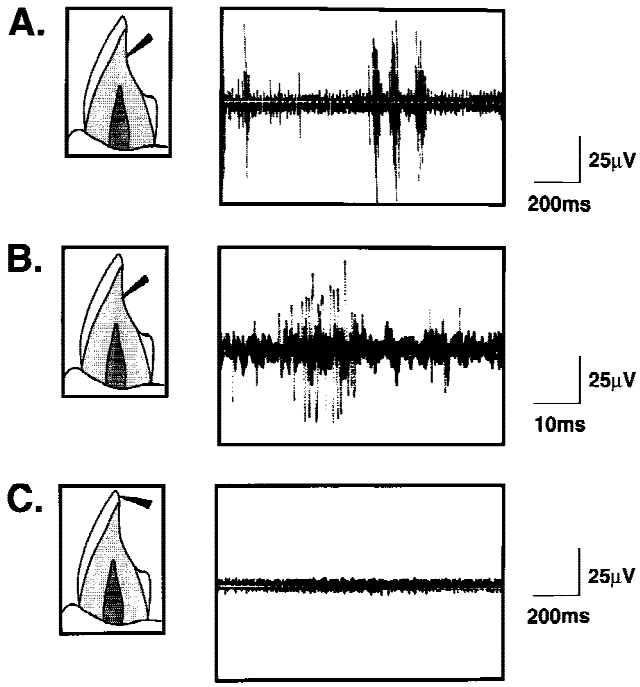

Fig. 2. Intradental nerve responses to mechanical stimulation of dentine with a glass probe (see inset diagrams). **A:** Bursts of intradental nerve activity generated by repeated mechanical stimulation of dentine. **B:** A burst of action potentials recorded at a faster sweep speed to resolve details of individual spikes. **C:** No activity was evoked when the probe was applied to enamel at the tip of the tooth.

four prelasing responses (Fig. 3, upper row) were respectively 19.7, 17.6, 8.7, and 18.9 V.s (mean, 16.2 V.s). After the first bout of lasing (0.6 W for 30 seconds) responses to mechanical stimulation showed more variability. The mean of the five responses after lasing was 13.9 V.s. (86% of the prelasing control value). Subsequent bouts of lasing caused further attenuation of the intradental nerve responses, and by the end of the experiment, the response to mechanical stimulation was greatly reduced. Figure 4 shows records of responses to mechanical stimulation of dentine obtained at the start of an experiment before any lasing and at the end of an experiment, after the tooth had been subjected to several bouts of laser irradiation. Some dentine preparations were also tested with hypertonic NaCl solutions, but these stimuli rarely produced any intradental nerve responses, probably because the remaining dentine thickness was too great. When present, the responses to NaCl solutions were affected by laser irradiation in the same manner as the responses to mechanical stimuli.

Figure 5 summarises the effects of lasing dentine on the responses to mechanical stimulation. The abscissa displays laser power, and the ordinate shows normalised intradental nerve responses expressed as a percentage of the control values. The open circles indicate the effects of single (30 seconds) bouts of lasing on responses from individual tooth preparations, and the filled circles represent the mean values for each laser power level. The effects of 60-second applications of the HeNe aiming beam alone are depicted on the graph as triangles and have been arbitrarily placed just above 0 W power.

The effect of lasing varied between preparations, but a general trend was evident by the progressive decline in the mean normalised postlasing responses as laser power was increased (Fig. 5, filled circles). Irradiation with the HeNe aiming beam and pulsed Nd:YAG irradiation at 0.3 W (plus HeNe) had no significant effect on intradental nerve responses which were, respectively, 104 ± 10% and 100 ± 13% of the prelasing controls. The effects of lasing at 0.6–1.5 W power varied between individual preparations and lasing either increased or decreased the responses to subsequent mechanical stimulation. With power levels of 2.0 W and above, intradental nerve responses were generally suppressed (Fig. 5). Analysis with the Kruskal-Wallis test revealed significant differences in the intradental nerve responses to mechanical stimulation after 30 seconds lasing at different power levels ($P = 0.02$). Intergroup comparisons with the Mann-Whitney test indicated that the responses were significantly depressed by lasing compared with controls only at 2 W ($P = 0.014$) and 3 W ($P = 0.01$).

Recovery of responsiveness after lasing varied between preparations. Some evidence of the extent of recovery can be seen in Figure 3. Here, the responses to repeated mechanical stimulation were generally suppressed after the second bout of lasing. In this preparation, further bouts of lasing (lower trace) greatly reduced the response to probing.

Laser-Induced Nerve Activity

The configuration of the dentine electrodes allowed recording to be continued during lasing, and intradental nerve activity was often generated during lasing in the absence of any other stimuli. Figure 6 shows laser-induced activity recorded in one preparation during sequential lasing at different power levels. In this tooth, lasing at 60 mJ, 10 pps evoked no intradental nerve ac-

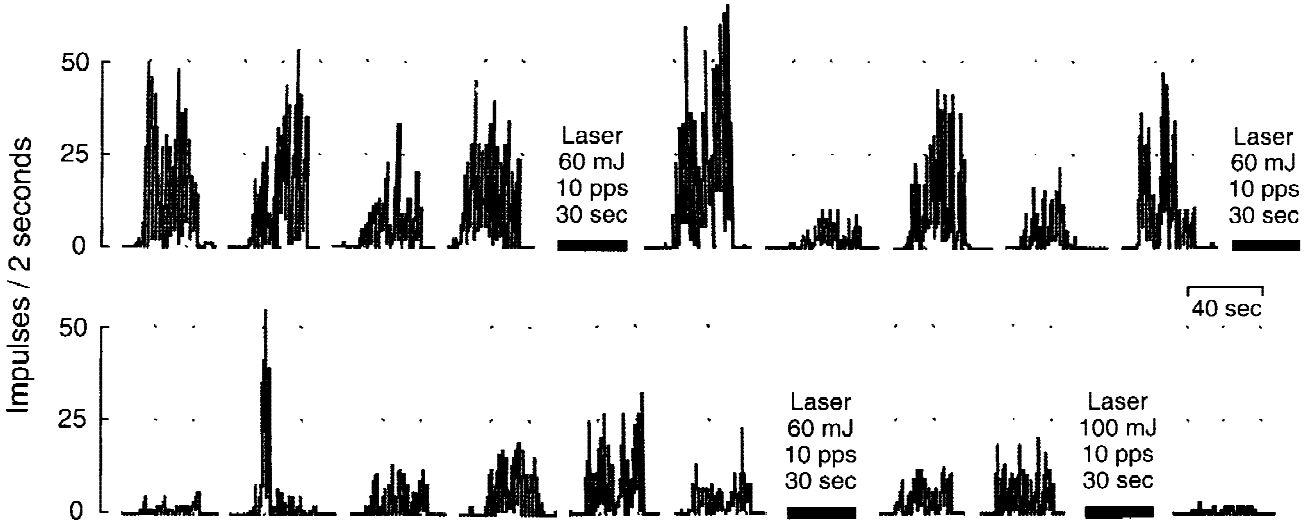

Fig. 3. Ratemeter records of intradental nerve activity generated by 30-second periods of mechanical stimulation. The stimulations were repeated at 5-minute intervals. In this experiment, dentine was lased four times at the points indicated by the black horizontal bars.

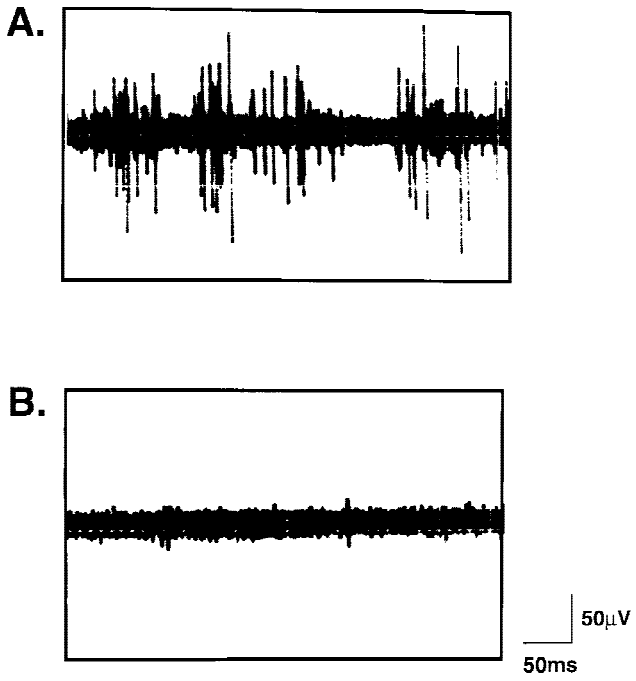

Fig. 4. Intradental nerve responses to mechanical stimulation of dentine. **A:** Before lasing. **B:** After two 30-second bouts of lasing at 0.6 W and 1.0 W.

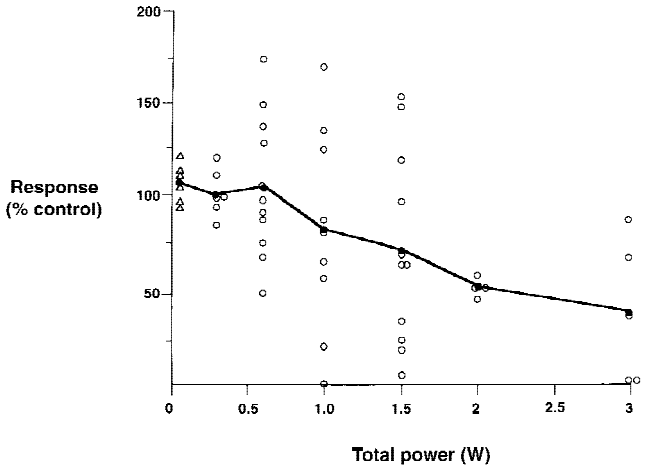

Fig. 5. Summary of the effects of irradiation at different laser power levels on the average response to 30-second mechanical stimulation. Triangles represent irradiation by the HeNe laser aiming beam alone. Circles represent responses after exposure to pulsed Nd:YAG laser plus HeNe irradiation: open symbols represent the mean effect of one laser exposure in one preparation, and the black circles represent the overall means for each power of laser exposure.

tivity (Fig. 6A). However, during lasing at 100 mJ, 10 pps (1.0 W power, Fig. 6B) and 100 mJ at 15 pps (1.5 W power, Fig. 6C), there was clear evidence of intradental nerve firing, which terminated soon after the lasing stopped. Finally, when the lasing at 1.5 W was repeated (Fig. 6D), no laser-induced activity was seen. We observed a trend in the incidence of laser-induced activity for various laser doses (Table 1). No laser-induced nerve activity was ever registered during lasing with the HeNe alone nor with the Nd:YAG at 0.3 W. The highest incidence of laser-induced nerve activity was with 1.0 W power, which evoked action potential discharges on seven of nine presen-

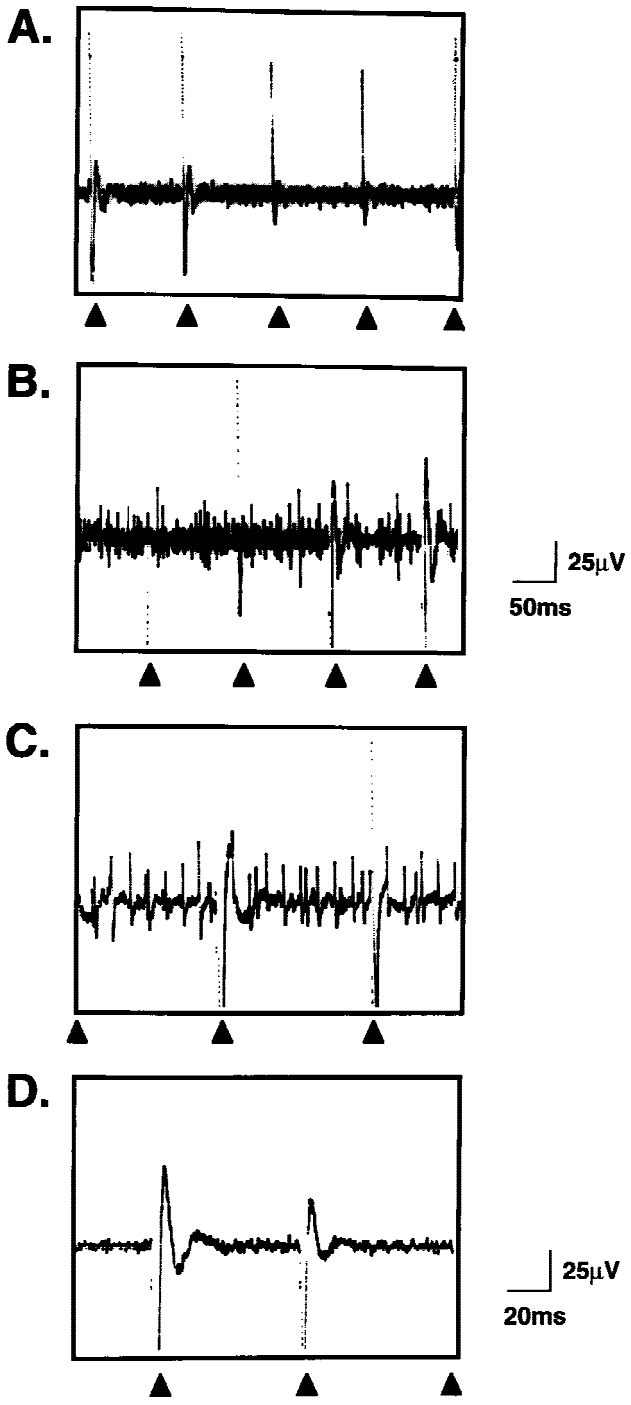

Fig. 6. Intradental nerve activity induced during lasing in one preparation. The large deflections (marked by arrowheads) are artefacts produced by the laser output pulses. **A:** 60 mJ, 10 pps (0.6 W). **B:** 100 mJ, 10 pps (1.0 W). **C,D:** 100 mJ, 15 pps (1.5 W). Note different time scales for records A,B and for C,D.

TABLE 1. Incidence of Intradental Nerve Activity Induced During Lasing

Nd:YAG laser settings (excludes HeNe control)	Power output (W)	Incidence of nerve activity	% incidence
Control (HeNe, 60 sec)	0 (nominal)	0/6	0
30 mJ, 10 pps, 30 sec	0.3	0/6	0
60 mJ, 10 pps, 30 sec	0.6	2/11	18
100 mJ, 10 pps, 30 sec	1.0	7/9	78
100 mJ, 15 pps, 30 sec	1.5	4/11	36
133 mJ, 15 pps, 30 sec	2.0	1/4	25
150 mJ, 20 pps, 30 sec	3.0	0/4	0

tations (78%). At power levels >1.0 W, the incidence of laser-induced activity declined.

DISCUSSION

Pulsed Nd:YAG irradiation is reported to be effective for treating "hypersensitive" dentine [2,3]. The mechanism of this desensitising effect is not certain, and the present experiments aimed to investigate how lasing might affect the sensitivity of laser-irradiated dentine to physiological stimulation. A mechanical stimulus was chosen because (1) this is a significant cause of pain amongst patients with "hypersensitive" dentine [19], (2) such stimuli are often used in the evaluation of desensitising treatments [2,3,20,21], and (3) this mode of stimulation is suitable for use with this experimental system [22].

It was found that irradiation with a HeNe laser alone or with the Nd:YAG laser at 0.3 W power produced no discernible effects on intradental nerve responses to mechanical stimulation of dentine. This result is not consistent with the clinical trial findings of Gelskey et al. [3], who reported that HeNe lasing reduced dentine sensitivity to air by 63% and to mechanical stimulation by 61% over a 3-month period; treatment with combined HeNe and Nd:YAG laser (up to 100 mJ, 10 pps) reduced sensitivity to air by 58% and to mechanical stimulation by 61%. However, our findings agree with reports that the HeNe laser had little effect on the frog sciatic nerve compound action potential [23]. Our results also support Wilder-Smith's conclusion [24], that HeNe laser irradiation alone was not an effective treatment for a variety of oral conditions, including "hypersensitive" dentine, gingivitis, and herpes labialis.

Irradiation with the Nd:YAG laser at more than or equal to 2.0 W power suppressed intradental nerve responses to mechanical stimula-

tion. The reduced intradental nerve responsiveness after lasing of dentine could be due to several factors, such as tubule occlusion, interference with the hydrodynamic transduction mechanism by which stimulus-induced fluid flow in dentinal tubules is converted into nerve activity or through a direct action on the nerves (e.g., by increased pulpal temperature). The present experiments do not permit us to distinguish between these possibilities. If the desensitising effects were due to occlusion of the outer ends of the dentinal tubules, further acid etching might be expected to restore responsiveness, but we found no evidence of this. Lan and Liu [5] reported that Nd:YAG laser treatment (at 30 mJ, 10 pps for 2 minutes) occluded dentinal tubule orifices. This finding would be expected to reduce dentine sensitivity to mechanical stimulation. We found no evidence that lasing at such energy levels (for 2 × 30 seconds) had any effect on intradental nerve responses to mechanical stimulation of dentine.

Laser-induced depression of intradental nerve excitability was to some extent reversible. Recovery of responsiveness was less complete after lasing at powers more than or equal to 2 W. Variations in the degree of recovery could be due to differences in the individual preparations, for example in the area of dentine exposed or thickness of dentine remaining over the pulp. In ferret canine teeth, the coronal dentine is approximately 0.6 mm thick, and there was little scope to vary dentine thickness in a systematic manner. Instead, we endeavoured to remove as little dentine as possible. We did not routinely measure the dentine thickness in the experiments, but some teeth were removed and examined microscopically. In the specimens examined, the amount of dentine remaining over the pulp at the base of the prepared cavity was in the range 0.35–0.60 mm.

The responses to mechanical stimulation were either suppressed or enhanced after lasing at powers in the range 0.6–1.5 W (Fig. 5). Some degree of variability is inevitable with the mode of stimulation used, but the method can achieve an acceptable degree of reproducibility as seen by the relatively small scatter of the responses after irradiation by the HeNe alone and Nd:YAG laser at 0.3 W (Fig. 5). Certainly, the variability of intradental nerve responses was often greater postlasing than prelasing (see for example, the upper tracings in Fig. 3). The increased responsiveness observed in some preparations after lasing at 0.6–1.5 W could be due to thermally-induced pulpal inflammation. Nd:YAG laser light is widely scattered in dentine and only about 1% is directly transmitted to the pulp [10]. As power levels increase, more energy reaches the inner dentine and is absorbed, causing increased hard tissue heating and consequently a rise in temperature within the pulp chamber. Any pulpal effects of lasing probably arise from this laser-induced heating of the dentine, rather than by direct irradiation of the tooth pulp. The temperature increases depend on the lasing parameters and the dentine thickness. White et al. [9] reported that lasing rat teeth for 30 seconds at 0.7 W power could raise pulp temperature by 6° C when dentine thickness was 2 mm; but when the dentine thickness was 0.2 mm, the pulp temperature rise was 43° C. The latter dimensions are more comparable to the ferret teeth used in the present experiments. However, the present experiments were carried out in vivo, and the Ringer's solution in the recording assembly may have lessened any temperature rises. It was not feasible to measure pulp temperature during the experiments, because insertion of a thermocouple into the pulp would inevitably damage the pulp nerves and, therefore, compromise the recordings.

Intradental nerve activity often occurred during lasing (Fig. 6; Table 1). The occurrence of laser-induced intradental nerve firing was related to the intensity of radiation, and was not evident during lasing at low power levels (<0.6 W). This activity was not obviously synchronised to the laser pulses but appeared as high frequency bursts of action potentials involving small groups of nerves. This finding is consistent with reports [25] that laser irradiation of cat teeth can evoke firing in single pulpal nerve axons. Lasing human teeth (at energies up to 100 mJ and 10 pps) can evoke painful sensations [3,4], and it is possible that the laser-induced activity observed in the present experiments is the neural correlate of laser-induced pain in humans.

The mechanism of laser-induced activity is not clear. The neural activity could arise from laser-induced changes in tubular fluid flow or a direct stimulation of pulpal nerves. As yet, there is no information on the effects of lasing on dentinal fluid flow rates. However, lasing-induced temperature increases could cause direct excitation of the pulp nerves. Heating teeth can evoke increased firing in intradental nerves [26]. It is not yet known whether this can occur with lasing. It may be of relevance that we failed to observe any laser-induced neural activity when lasing isolated nerves, either directly [27] or when the nerves

were placed within a tooth segment to mimic a pulp chamber [28].

There is little information on the effects of lasing on intradental nerve responsiveness to physiological stimulation. Our findings are consistent with the results of Tokita [14], who applied pulsed Nd:YAG laser irradiation to the enamel of cat canine teeth. He reported that lasing could reduce the excitability of intradental nerves to both mechanical and osmotic stimulation of dentine. He found that the laser-induced pulpal effects were largely confined to the irradiated coronal part of the tooth, and he reported histological evidence of localised tissue damage in the pulp with power levels as low as 0.6 W. However, although pulsed Nd:YAG laser irradiation of vital teeth could depress intradental nerve activity, it can also damage the pulp [25].

The lasing parameters used in this study were comparable to those used for treating hypersensitive dentine in human subjects [2,3]. The effects of a given level of laser irradiation are likely to be greater in ferret teeth than in human teeth, because ferret teeth are smaller and have less dentine (ca. 0.6 mm) over the pulp compared with human teeth where the dentine is 2-3 mm thick. Brief (30 second) exposures to low power levels (<0.6 W) are reported to have no adverse effect on pulp microstructure in rat, monkey and human teeth [10]. The present investigation showed that such low levels of laser irradiation (with either the HeNe or Nd:YAG lasers) applied to dentine had little or no effect on ferret intradental nerve excitability. However, lasing at 1 W average power can suppress intradental nerve excitability to mechanical stimulation of dentine. Lasing at powers more than or equal to 2 W caused irreversible depression of intradental nerve responses and has been reported to cause pulpal damage in monkey or human teeth, especially when the remaining dentine thickness is less than 2 mm [10]. Further research is necessary to establish why lasing dentine at power levels in the range 0.6–1.5 W could have such variable effects on intradental nerve excitability.

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